Contaminant Stressors

Background

The Stanislaus River, between Goodwin Dam and Caswell State Park, has been identified as being impaired on the USEPA Clean Water Act Section 303(d) list for not meeting water quality standards since the early 1990s. The pollutants or stressors that have been identified to cause the impairments are: diazinon, chlorpyriphos, Class A pesticides (e.g., organochlorines, DDT, and legacy pesticides), unknown toxicity, mercury, and temperature (USEPA 2011). In addition, mercury and selenium have been identified as impairing beneficial uses in the San Joaquin River, the Delta, and the San Francisco Bay, which are downstream salmonid rearing and migratory habitats (SWRCB 2010; USEPA 2011). Beneficial uses that are not being supported include: cold freshwater habitat; migration; spawning, reproduction and early development; and warm freshwater habitat. Some other contaminants that were evaluated, but they were found not to exceed water quality standards included ammonia, arsenic, cadmium, and nickel (SWRCB 2010).

The large majority of currently available spawning habitat and subsequent rearing habitat in the Stanislaus River is below Knights Ferry (ESA 2013), and this reach coincides with increased amounts of anthropogenic disturbances, primarily agricultural and urban development. In a review of toxicity monitoring data conducted in California, Anderson and others (2011) found that sites located near agriculture and urban areas had statistically greater occurrences of toxicity in water and sediment samples than near undeveloped areas. In all, 51% and 45% of the streams, rivers, canals, and lakes monitored from 2001 to 2010 had some toxicity in the water column and sediment, respectively. Toxicological effects can range from sublethal endpoints to full organism mortality. Using correlation analyses and toxicity identification evaluations, Anderson et al. (2011) determined that the vast majority of toxicity was cause by pesticides (e.g., insecticides, herbicides, and fungicides). However, pesticides were not the cause of all toxicity, and some other contaminants that were identified included metals and ammonia.

The Central Valley Regional Water Quality Control Board (Central Valley Water Board) has recently developed a control program and adopted water quality objectives for diazinon and chlorpyriphos in the Central Valley (CVRWQCB 2014), so the implementation of the program should reduce the adverse impacts of these two constituents. However, the use of organophosphate pesticides like diazinon and chlorpyriphos have decline in California since the mid-1990s, and USEPA actions resulted in the phase out of these two pesticides for urban use in the early 2000s (Spurlock and Lee 2008). Much of the pesticide use has shifted to pyrethroids, especially for urban use, and in 2006 pyrethroids accounted for greater than 40% of the insecticide registrations in California. Pyrethroids have been identified as causing much of the surface water and sediment toxicity in California (Anderson et al. 2011). More recently, the use of the systemic pesticides neonicotinoids has increased, and their use has been implicated in global declines of some wildlife (Gibbons et al. 2014; Mason et al. 2012). Current use pesticides are ever changing, and this makes it difficult for regulatory agencies to control the adverse effects that these contaminants create.

Mercury and selenium both occur naturally in the environment; however, anthropogenic activities have resulted in elevated concentrations in surface waters that are a detriment to aquatic life. For centuries, the smelting of large quantities of ore has contributed to the emissions of trace metals worldwide (Nriagu 1996). Recently, mercury water quality impairments in California have been linked to local and international industrial emissions (SFEI 2001; USEPA 2008). Extensive historical mining in California contributed to heavy metal emissions, as well abandoned mine waste material continues to pollute Central Valley water bodies (Alpers and Hunerlach 2000; Domagalski 2001; USEPA 2006). Oil refining and agricultural irrigation have contributed to selenium contamination in the San Francisco Bay and the San Joaquin River watershed, respectively (McCarthy and Grober 2001; Presser and Luoma 2006 and 2013). In addition, urban storm water runoff has been shown to be a major source of metals to California surface waters (CRWQCBSDR 2007; SFBRWQCB 2007; TDC 2004).

The following sub-sections will describe the three major contaminants (pesticides[1], mercury, and selenium) that have been identified as impairing beneficial uses in the Stanislaus River and downstream migratory corridor. The descriptions of each contaminant will follow similar formats. First, general background on the contaminant and the toxicological effects of each contaminant to fish, with emphasis on salmonids where available, will be described in the text. Second, the environmental objectives (e.g., benchmark concentrations, exposures, etc.) of each contaminant predicted to be necessary to attain the biological objectives will be summarized from the available literature, current or proposed water quality criteria or objectives, etc. Finally, the text will describe the predicted current conditions of the contaminants, and overall the overall risks of salmonid populations to exposures to the contaminants in the Stanislaus River and downstream watershed.

Some of the identified contaminants have associated USEPA promulgated numeric aquatic life water quality or human health criteria (California Toxics Rule [CTR], 40 CFR Part 131), as well as each may have Regional Board specific water quality objectives. Unfortunately, most current use pesticides do not have promulgated water quality criteria or objectives. Additionally, the CTR criteria were developed to protect for human health or against short-term (4-day) effects on aquatic life, and these criteria may not be protective of long-term (e.g., weeks, months, and years) adverse impacts on salmonids and other wildlife. For example, the evaluation for the Sacramento-San Joaquin Delta Estuary Total Maximum Daily Load (TMDL) for Methylmercury determined that even though the CTR criterion for mercury is never exceeded in the Delta, fish tissue mercury concentrations are a threat to threatened and endangered wildlife species and humans that consume Delta fish (Wood et al. 2010). As well, many of the toxicological studies to be discussed later have observed adverse effects to salmonids below established water quality criteria.

Pesticides

Fish are not the target organisms of the pesticides; however, pesticides have been found to cause adverse impacts to fish in surface waters. For example, in a review of Central Valley toxicity data, Markiewicz and others (2012) found that the fish species tests, *Pimephales promelas*, had a higher frequency of toxicity than the other species, *Ceriodaphnia dubia* (invertebrate) and *Selenastrum*

[1] The pesticide section will include a discussion on copper effects because copper is widely used as pesticide (e.g., fungicide and antifouling paint).

capricornutum (algal). Samples were toxic to fish in 62% of the tests versus 49% for invertebrates and 40% for algae. Similar to the statewide survey of Anderson and others (2011), pesticides were found to be the primary cause of toxicity in the Central Valley (Markiewicz et al. 2012). Importantly, salmonids generally tend to be more sensitive to chemical stressors than many other species of fish; and, if other freshwater fish are killed by use of pesticides, then it is likely that salmonids have also died (NMFS 2012b).

Moreover, the life history strategies salmonids evolved to rely on exposes them to higher risks from contaminants. For example, juvenile salmonids typically occupy and rely on shallow freshwater habitats (e.g., floodplains, off-channel, and low flow alcoves) during critical rearing and migratory life history periods. These near-shore, low flow habitats are expected to have higher pesticide loading and concentrations, which subject developing salmonids to higher exposures to pesticides in their preferred habitats (NMFS 2008, 2009c, and 2011c). Even if salmonids can avoid the elevated concentrations of contaminants in these areas, salmonids may be adversely impacted by not benefitting from the uses these habitats provide (e.g., food and cover).

Typically, adult organisms will have a lower risk of mortality to contaminants than the sensitive larval fish used for toxicity tests. As a result, toxicity tests with larval fish could overestimate the mortality that might occur to adult salmonids. However, pre-spawn adult salmonids are likely less tolerant to chemical stressors because they have used most of their accumulated fat stores for gamete production (NMFS 2008, 2010, and 2013b). It is probable that the some pre-spawn returning adults will die as a result of short-term exposures to pesticides, especially when subjected to additional stressors like elevated temperatures. Additionally, pre-spawn mortality can be cause by other contaminants. For example, metals and petroleum hydrocarbons likely contributed to pre-spawn mortality of Coho salmon in urban streams in Washington State (Scholz et al. 2011). Pre-spawn mortality is a particularly important factor in the recovery of salmonid populations with low abundance because every adult is crucial to the population's viability (NMFS 2013b).

While direct mortality is an obvious detriment to salmonid populations, many sublethal effects of pesticide can also contribute to population declines. Sublethal toxicant exposure often eliminates the performance of fish behaviors, such as predator avoidance, orientation, reproduction, kin recognition, etc. that are essential to fitness and survival in natural ecosystems (Potter and Dare 2003; Scott and Sloman 2004). The most commonly observed links with behavioral disruption include cholinesterase (ChE) inhibition, altered brain neurotransmitter levels, sensory deprivation, and impaired gonadal or thyroid hormone levels (Scott and Sloman 2004). For example, Scholz and others (2000) concluded that olfactory disruption by anti-cholinesterase neurotoxins reduced Chinook salmon anti-predator responses from short-term, sublethal exposures to diazinon. As well, they also concluded that 24-hour exposures to diazinon likely increased the straying of the adult hatchery Chinook salmon over the control group. Furthermore, juvenile salmonids exposed to pesticides during development may fail to imprint to their natal waters, which can lead to increased adulthood straying (NMFS 2009c).

Additional evidence of the sublethal effects of pesticides on fish populations have been demonstrated though reproduction experiments. For example, the pyrethroid insecticide cypermethrin inhibited male

Atlantic salmon from detecting and responding to the reproduction priming pheromone prostaglandin, which is released by ovulating females (Moore and Waring 2001). The males exposed to cypermethrin did not respond to prostaglandin with the expected increased levels of plasma sex steroids and expressible milt. In addition, zebrafish exposed to low concentrations (96-hour LC5) of deltamethrin and Achook (a synthetic pyrethroid and a neem based pesticide, respectively) resulted in significant reductions (54% and 18%, respectively) in female fecundity when compared to the controls (Sharma and Ansari 2010). Additionally, both of the studies found that exposures to pesticides decreased the abundance of hatchlings. The percentage of unhatched fertilized eggs increased in adult zebrafish exposures, and the number of unfertilized eggs increased in salmon egg and milt exposures (Sharma and Ansari 2010; Moore and Waring 2001). Furthermore, the disruption of spawning synchronization could also result in an increase in the number of unfertilized eggs (NMFS 2009c).

Herbicide pesticides also have been shown to reduce fish's ability to perform necessary physiological activities. For example, Waring and Moore (1996) observed that concentrations of the herbicide atrazine that showed no lethal effects to Atlantic salmon in freshwater resulted in physiological stress and increased mortality once the fish were exposed to seawater. Subsequent investigations determined that sublethal concentrations of atrazine can reduce $Na^+ K^+$ ATPase activity and the ability of salmon to osmoregulate (Moore and Fewings 2003). Nieves-Puigdoller and others (2007) found similar disruptions in osmoregulation as well as other endocrine disruption, however at higher concentrations of atrazine. Other investigations have concluded that another herbicide, trifluralin, can cause vertebral deformities, which would likely also result in the eventual mortality from predators or reduced prey capture (NMFS 2012b). Because pesticides are developed and used for multiple target organisms (e.g., plants, invertebrates, and vertebrates), their mechanisms of action are very diverse. This results in a multitude of ways that pesticides can affect salmonid physiology, biochemistry, and behavior, and subsequently, many different life stages of salmonids can be adversely impacted.

Copper compounds are also often used as herbicides in addition to other types of pesticides, and copper is one of the most widely applied pesticides in the Central Valley (Johnson et al. 2010). Additionally, copper is a naturally occurring trace element, and non-pesticide related anthropogenic activities have increased copper pollution to surface waters. For example, other sources of copper to surface waters include: urban runoff (e.g., vehicle brake pads, architectural features, and industrial uses), mining waste, soil erosion, etc. (CVRWCB 2002; TDC 2004). Extreme cases of copper and other heavy metal contamination resulted in acid mine drainage that contributed to fish kills and significant declines in Chinook salmon and steelhead populations in the Sacramento River from the 1960s to the 1980s (CVRWQCB 2002). Heavy metal pollution from the Iron Mountain Mine to the Sacramento River contributed to the listing of winter-run Chinook salmon as endangered (CVRWQCB 2002).

Current copper pollution from pesticides and urban runoff are not as extreme as the Iron Mountain Mine example; however, low levels of copper can have adverse effects on salmonids, other fish, invertebrates, and algae (Hetch et al. 2007; USEPA 2007). The most studied toxicity pathway of copper is its ability to disrupt ATP-driven pumps and ion channels, which results in impaired osmoregulation and ion regulation in gills (Kiaune and Singhasemanon 2011). However, fish sensory systems are likely the most sensitive to sub-lethal copper toxicity. For example, low-level copper exposures have been shown

to disrupt olfactory receptor neurons and lateral line mechanosensory neurons in fish (Hansen et al. 1999a; Hecht et al. 2007; Linbo et al. 2009; McIntyre et al. 2008; Sandahl et al. 2007). In addition, these copper exposures resulted in measured behavior alterations (e.g., predator avoidance response, contaminant avoidance, and swimming) in Chinook salmon and rainbow trout that could result in reduced growth, survivability, and reproduction in salmonid populations (Hansen et al. 1999b; Sandahl et al. 2007).

Indirect Effects

Salmonid populations can also be adversely impacted indirectly by pesticides acting upon their target species. For example, herbicides and insecticides target the food web organisms that the salmonids depend on during rearing and migration. In addition, pesticides in the aquatic environment can shift algal or invertebrate communities to ones that are less nutritious or preferable to salmonids. Modifications to prey and prey food sources can have noticeable effects on fish populations (NMFS 2012b). Reduced food for developing salmonids will result in greater competition, reduced fish growth, and possible starvation during critical life stages (NMFS 2008). Other possible indirect impacts to salmonid populations include the destruction of riparian vegetation (NMFS 2012b). Riparian vegetation is important for providing shade, stabilizing stream banks, and providing allochthonous inputs that are important to maintaining salmonid ecosystems.

Population Level Effects

It is very difficult to quantify actual impacts that pesticide stressors have on salmonid populations because the effects can be direct or indirect, lethal or sublethal, long-term or short-term. To determine the possible combined effects that pesticides might have on salmon populations, researchers at the Northwest Fisheries Science Center used models to predict the effects of ChE inhibitors on anadromous Chinook salmon populations in the western United States (Baldwin et al. 2009; Macneale et al. 2014). They linked ChE activity to the somatic growth of subyearling Chinook salmon using a series of linear relationships (e.g., linked brain enzyme activity to feeding behavior, feeding behavior to food uptake, and food uptake to somatic growth). In addition, the researchers predicted the reduction in Chinook salmon growth due to reduced prey as a result of invertebrate exposure to pesticides. The predicted size of Chinook salmon at ocean entry is used to predict ocean survival, and then subsequent population growth.

The model results indicated that short-term exposures that were representative of real-world seasonal use patterns were enough to reduce the growth and size of juvenile Chinook salmon at the time of ocean entry. Consequently, the reduced size at ocean entry was enough to reduce the survival of individuals, which would, over successive years, reduce the intrinsic productivity of the population. For example, a four-day exposure to an organophosphate pesticide at a level that would produce a 50% reduction in ChE activity would result in a 6% decrease in the intrinsic population growth rate (Baldwin et al. 2009). Furthermore, the model estimated that if similar conditions continued for 20 years, then the exposed population spawner abundance would be only 27% of the unexposed spawner abundance. Macneale et al. (2014) evaluated additional pesticide classes (e.g., carbamates), exposure durations, and exposure frequencies. Overall, the magnitude of the responses indicates that common pesticides may significantly limit the conservation and recovery of threatened and endangered species in California

(Baldwin et al. 2009).

Unfortunately, the models only evaluated the direct and indirect effects of single pesticide exposures at a time, and they did not incorporate possible interactions of multiple pesticides, other environmental stressors (e.g., reduced habitat and sub-optimal temperatures), or other contaminants. Different pesticides can work additively to cause a toxic effect, and other contaminants and stressors can influence pesticides' effectiveness, as well. For example, through transcriptional assays Hasenbein et al. (2014) determined that ammonia likely enhanced the effect of multiple-contaminant exposures to Delta smelt. Similarly, concurrent exposure of salmonids to copper and olfactory inhibitory pesticides could result in toxicological effects, even if both are at concentrations that would not elicit a response in isolation. Furthermore, many pesticides have been found to be able to work synergistically to cause toxicity to salmonids that is multiplicative and not just additive (Laetz et al. 2009). Current estimates of the effects of pesticides on salmonids may underestimate the true responses of salmonid populations in surface waters (Baldwin et al. 2009).

These additive and synergistic effects from multiple contaminants are true concerns for aquatic environments. For example, in the National Water-Quality Assessment (NAWQA) Program's monitoring of pesticides, they found that more than 90% of the streams located in developed areas contained two or more pesticides or degradates (Gilliom et al. 2006). Furthermore, more than 50% of the streams had five or more pesticides or degradates, and the concentrations of the degradates were often higher than that of the parent pesticide. The degradate forms can be less toxic than the parent pesticide; however, some degradates have been found to be as toxic or more toxic than the parent (Gilliom et al. 2006). In addition, pesticide products typically contain additional chemicals like adjuvants, surfactants, and solvents. These chemicals are labeled as inert ingredients, but they increase the effectiveness of the active ingredients and can be toxic to non-target species (Beggel et al. 2010; Cox and Surgan 2006; Scholz et al. 2012). Very little is known about the fate of these "inert" labeled ingredients once they are in surface waters and their possible impacts on salmonid populations.

Pesticide Objectives

Numeric water quality objectives have not been established for vast majority of current use pesticides in the Central Valley. Table 1 presents the pesticides that have adopted numeric water quality objectives in the Sacramento and San Joaquin River Basins Water Quality Control Plan (Basin Plan) and the proposed water quality objectives for pyrethroid pesticides (CVRWQCB 2011; CVRWQCB 2014a; CVRWQCB 2014b). The Basin Plan primarily relies on narrative water quality objectives for pesticides and toxicity to protect aquatic life beneficial uses. For example, for pesticides the Basin Plan states "No individual pesticide or combination of pesticides shall be present in concentrations that adversely affect beneficial uses...", and for toxicity it states "All waters shall be maintained free of toxic substances in concentrations that produce detrimental physiological responses in human, plant, animal, or aquatic life..." Unfortunately, the narrative criteria make it difficult to assess the possible environmental impacts of known pesticide concentrations in the river without bioassays, bioassessment investigations, etc.

Table 1

Central Valley Regional Water Quality Control Board Adopted and Proposed Water Quality Objectives for Current Use Pesticides

Pesticide	Acute (µg/L)	Chronic (µg/L)
Adopted Water Quality Objectives[1]		
Diazinon	0.16	0.1
Chlorpyriphos	0.025	0.015
Carbofuran	40	40
Simazine	4	4
Thiobencarb	1	1
Pentachlorophenol	5.3	4
Copper	5.7	4.1
Proposed Water Quality Objectives[2]		
Bifenthrin	0.004	0.0006
Cyfluthrin	0.0003	0.00005
Lambda-Cyhalothrin	0.001	0.0005
Cypermethrin	0.001	0.0002
Esfenvalerate	0.02	0.003
Permethrin	0.01	0.002

Notes:
[1]CVRWQCB 2011
[2]Proposed water quality objectives for the Central Valley Pyrethroid Pesticides TMDL and Basin Plan Amendment (CVRWQCB 2014b).

USEPA Office of Pesticide Programs (OPP) develops aquatic toxicity benchmarks for use in risk assessment and pesticide registration decisions under the Federal Insecticide, Fungicide, and Rodenticide Act (USEPA 2004). OPP has developed aquatic life benchmarks for over 400 registered pesticides. Table 2 presents the benchmarks for the 40 pesticides that are predicted to pose the greatest risks in the Central Valley (Lu and Davis 2009; Hoogeweg et al. 2011). Included in Table 2 are the benchmarks for the protection of the critical habitat for listed species, which includes an additional safety factor (USEPA 2004). The aquatic life benchmarks can be used for initial environmental assessments; however, a more detailed evaluation or site-specific evaluations may determine that the aquatic life benchmarks are not protective of the most sensitive species. For example, a comparison between the OPP benchmarks (Table 2) and the established or proposed water quality objectives (Table 1) shows that all but one of the water quality objectives predicts that a lower concentration than the OPP benchmarks is necessary to protect beneficial uses. Attaining the lower of either the aquatic life benchmarks or the water quality objectives should reasonably allow for the protection of salmonid species as well as their habitat.

Table 2

USEPA Office of Pesticide Programs' Aquatic-Life Benchmarks for the 40 Pesticides That Pose the Greatest Risk in the Central Valley Region

Pesticide	Pesticide Type	Acute Benchmark (µg/L)	Endangered and Threatened Acute Benchmark (µg/L)	Chronic Benchmark (µg/L)	Source of Acute/ Chronic Value[1]
Abamectin	Insecticide	0.17	0.017	0.006	IA/IC
Bifenthrin	Insecticide	0.075	0.0075	0.0013	FA/IC
Bromacil	Herbicide	6.8	0.68	3000	AA/FC
Captan	Fungicide	13.1	1.31	16.5	FA/FC
Carbaryl	Insecticide	0.85	0.085	0.5	IA/IC
Chlorothalonil	Fungicide	1.8	0.18	0.6	IA/IC
Chlorpyrifos	Insecticide	0.05	0.005	0.04	IA/IC
Clomazone	Herbicide	167	16.7	350	AA/FC
Copper hydroxide	Fungicide	5.9	0.59	4.3	IA/IC
Copper sulphide	Insecticide/Algaecide	5.9	0.59	4.3	IA/IC
Cyfluthrin	Insecticide	0.0125	0.00125	0.007	IA/IC
Cyhalofop butyl	Herbicide	245	24.5	134	FA/FC
Cypermethrin	Insecticide	0.195	0.0195	0.069	FA/IC
Deltamethrin	Insecticide	0.055	0.0055	0.0041	IA/IC
Diazinon	Insecticide	0.11	0.011	0.17	IA/IC
Dimethoate	Insecticide	21.5	2.15	0.5	IA/IC
Diuron	Herbicide	2.4	0.24	26	AA/FC
Esfenvalerate	Insecticide	0.025	0.0025	0.017	IA/IC
Hexazinone	Herbicide	7	0.7	17000	AA/FC
Imidacloprid	Insecticide	35	3.5	1.05	IA/IC
Indoxacarb	Insecticide	12	1.2	3.6	FA/IC
Lambda cyhalothrin	Insecticide	0.0035	0.00035	0.002	IA/IC
Malathion	Insecticide	0.3	0.03	0.035	IA/IC
Mancozeb	Fungicide	47	4.7	N/A	AA/na
Maneb	Fungicide	13.4	1.34	N/A	AA/na
Methomyl	Insecticide	2.5	0.25	0.7	IA/IC
(s)-Metolachlor	Herbicide	8	0.8	30	AA/FC
Naled	Insecticide	25	2.5	0.045	AA/IC
Oxyfluorfen	Herbicide	0.29	0.029	1.3	AA/FC
Paraquat	Herbicide	0.396	0.0396	N/A	AA/na
Pendimethalin	Herbicide	5.2	0.52	6.3	AA/FC
Permethrin	Insecticide	0.01	0.001	0.0014	IA/IC
Propanil	Herbicide	16	1.6	9.1	AA/FC
Propargite	Insecticide	37	3.7	9	IA/IC

Pyraclostrobin	Fungicide	0.0015	0.00015	0.002	FA/FC
Simazine	Herbicide	36	3.6	960	AA/FC
Thiobencarb	Herbicide	17	1.7	1	AA/IC
Tralomethrin	Insecticide	0.055	0.0055	0.0041	IA/IC
Trifluralin	Herbicide	7.52	0.752	1.14	AA/FC
Ziram	Fungicide	9.7	0.97	39	FA/IC

Notes:
Table modified from Hoogeweg et al. (2011). Aquatic-life benchmarks are used by the USEPA-OPP for risk assessments in the registration of pesticides. The entire list of nearly 500 pesticide benchmarks can be acquired at: http://www.epa.gov/oppefed1/ecorisk_ders/aquatic_life_benchmark.htm
[1]Identifies which taxa was the most sensitive to the pesticide from available toxicity evaluations: FA = fish acute; IA = invertebrate acute; AA = Algae Acute; FC = fish chronic; IC = invertebrate chronic; na = not available

The pesticide objectives were developed assuming organismal exposure to single pollutants. Additional considerations will be necessary, if multiple pesticides are present (e.g., additive toxicity equations). In addition, assessing the true impact on aquatic life may need to consider the bioavailability of the pesticides. For example, the majority of dissolved copper is likely bound as ligand complexes and largely not bioavailable. Consequently, copper, pesticides, and other metals toxicity evaluations should involve adjustments for site-specific conditions (e.g., hardness, biotic ligand models, or dissolved organic concentrations).

Pesticide Exposures to Salmonids

Pesticide applications are highly seasonal, and application timing varies by crop type, weather, and land use type. Subsequently, pesticide runoff and salmonid exposure to elevated concentrations of pesticides will also be seasonal and affected by other environmental conditions. Quantifying the concentrations of all the pesticides that salmonids are exposed to is difficult. For example, over 1000 pesticide chemicals were applied in California in 2012 (CDPR 2014). In addition, each commodity or crop type can have multiple pesticide chemicals that are applied to them (e.g., alfalfa crops were associated with greater than 200 pesticide chemicals). Performing chemical analyses, for all possible pesticides in the different reaches of the river where salmonids would be exposed, would not be cost feasible. Furthermore, current analytical methodologies do not allow for all pesticides to be detected at levels that may cause adverse effects to aquatic organisms. For instance, only recently have techniques been developed to reliably detect many pyrethroid pesticides in surface waters at concentrations near or below sensitive species' LC50's (Hladik et al. 2009; Mekebri 2011). Even still, LC50 values are concentrations where 50% of the organisms experience mortality. Sublethal effects are likely occurring to salmonid population even if the pesticides or mixtures of pesticides are not detected.

The current limitations of pesticide monitoring in surface waters has prompted the use of models to predict surface water pesticide concentrations and to assess pesticide risks to aquatic organisms. For example, in 2001 the NAWQA program developed a model, Watershed Regressions for Pesticides, to predict atrazine concentrations in national streams (USEPA River Reach File 1; Horn et al. 1994), and the program recently expanded the model to predict the concentrations of multiple pesticides (Stone et al. 2014). Similarly, the USEPA Office of Pesticide Programs uses various water exposure models to assess

the risk of pesticides to aquatic organisms and the environment (USEPA 2014a).

Hoogeweg et al. (2011) used modeling to quantify the spatial and temporal pesticide risks to threatened, endangered, and other species of concern in the Sacramento River, San Joaquin River, and San Francisco Bay-Delta watersheds. Chinook salmon (Sacramento winter-run, Central Valley spring-run, Central Valley fall-run, and Central Valley late fall-run) and steelhead were included on list of nine species of concern. They predicted the frequency that pesticides would exceed aquatic-life benchmarks and the co-occurrence of these exceedances with the species of concern. At least a portion of the Stanislaus River was identified as a "Potential Area of Concern" (i.e., a high frequency of both pesticide exceedances and species richness) in all months except August and November (Hoogeweg et al. 2011 [Figures 77 to 88]). However, individual species may still be at risk during these two months because the model does predict that benchmark exceedances would occur, on occasion, during these months.

The Hoogeweg et al. (2011) model allowed the determination of the magnitude of pesticide effects on Stanislaus River salmonids, and the relative risk of pesticide exposures by month and river reach (Figure 1 and Table 3). As mentioned earlier, limitations in monitoring and chemical analyses, the multitude of possible pesticide chemicals, etc. precludes the use of strict concentration limitations to evaluate overall pesticide impacts on salmonids throughout the Stanislaus River. In turn, current pesticide impacts to salmonid life stages in the Stanislaus River are based on the relative frequency of pesticides exceeding aquatic-life benchmarks. The target condition for pesticide impacts is zero to little frequency of benchmark exceedances (i.e., Bins 1 and 2 or less than 5% exceedance).

The Hoogeweg et al. (2011) model evaluated exposures to copper from pesticide specific sources; however, copper from other sources (e.g., vehicles or mining) were not assessed. Salmonid exposures to copper, regardless of the source, in the Stanislaus River and downstream migratory pathways can be evaluated by comparing water quality monitoring data to the copper benchmark for endangered and threatened species (0.59 µg/L, assuming a hardness of 40 mg/L). Average and maximum concentrations of dissolved copper in the Stanislaus River were 0.8 and 2.1 µg/L (n = 20), respectively, from 2000-2002 and 1.6 and 3.4 µg/L (N = 124), respectively, from 1993 to 2013 in the San Joaquin River from Vernalis to the Delta (CEDEN 2014a and 2015). Chinook salmon have been found to avoid dissolved copper concentrations of 0.7 µg/L, and rainbow trout were found to avoid 1.6 µg/L (Hansen et al. 1999b). In addition, Chinook salmon acclimated to 2 µg/L dissolved copper failed to avoid higher concentrations of copper (Hansen et al. 1999b). Similarly, sensory physiology and predator avoidance were both impaired in juvenile Coho at 2 µg/L copper (Sandahl et al. 2007). Copper concentrations in the Stanislaus River and in salmonid migratory pathways are at levels that may pose risks to salmonid populations.

Summary

Pesticides and copper have a high potential to greatly impact salmonid survival and population recovery. The diverse mechanisms of action of the different types of pesticides found in the aquatic environment have the ability to affect all the life stages of salmonids as well as the ecosystem that they rely on. However, measuring the true impacts of pesticides on salmonid populations is very difficult. As well, the magnitude of pesticide impacts compared to other possible stressors (e.g., temperature, reduced habitat, and predation) is unknown. All the stressors likely work in combination to reduce salmonid

fitness. Consequently, potential pesticide impacts should be considered with the other stressors for salmonid population recovery, especially in developed areas such as the California Central Valley.

Table 3
Categories of Predicted Pesticide Aquatic-life Benchmark Exceedances

Bin Category	Range of the Frequency of Benchmark Exceedances	Severity Ranking
1	0 - 0.017	A
2	0.018 - 0.055	A
3	0.056 - 0.1	B
4	0.101 - 0.153	B
5	0.154 - 0.206	B
6	0.207 - 0.303	B
7	0.304 - 0.447	B
8	0.448 - 0.5	C
9	0.501 - 0.589	C
10	0.59 - 0.994	C

Note:
Frequencies were calculated from the total number of predicted exceedance days for each month from 2000 to 2009. Any day that had at least one pesticide that exceeded benchmarks was counted as an exceedance day (adapted from Hoogeweg et al. 2011).

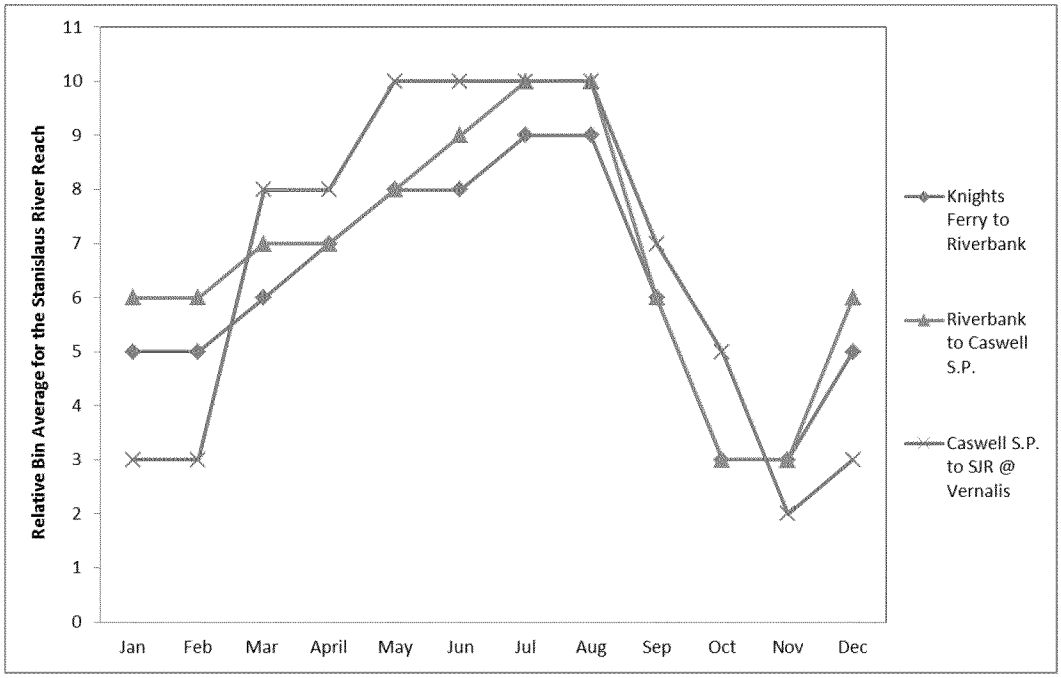

Figure 1

Relative Bin Value of Specified Stanislaus River Reaches by Month

Note: The values were derived from qualitative averaging of the frequency of benchmark exceedances model maps for years 2000 to 2009 in Hoogeweg and others (2011). Due to a lack of data, upstream of Knights Ferry in the Stanislaus River was not modeled.

Mercury

Mercury is a persistent and bioaccumulative toxic pollutant. Methylmercury is the most toxic form in the freshwater environment because it is the form most readily bioaccumulated in fish and through the food web (Wiener et al. 2003). For example, the proportion of mercury that exists as methylmercury generally increases with each level of the food chain, and methylmercury comprises 80% to 100% of the total mercury measured in fish tissue (Becker and Bigham 1995; Bloom 1992; Nichols et al. 1999; Slotton et al. 2004; Sveinsdottir and Mason 2005; Weiner et al. 2003). Fish can absorb mercury through their epidermis (gills, skin, etc.) directly from water; however, fish accumulate the majority (>85%) of their mercury through their diet in the form of methylmercury (Hall 1997; Weiner et al. 2003). There is evidence that methylmercury bioconcentrates (directly from water) in the laboratory (Fjeld et al. 1998; McKim et al. 1976); however, the minimum concentrations used in these dilution series exposures (160 and 30 ng/l, respectively) were greater than 25-fold higher than the maximum aqueous methylmercury concentrations found in Central Valley mainstem rivers (Foe et al. 2008). It is the result of bioaccumulation and subsequent biomagnification that methylmercury concentrations typically become elevated in fish, and fish in the higher tropic levels tend to have the highest concentrations.

Fish have evolved in environment that always contained mercury. Methylmercury is transported via the circulation system to all organs and tissue; however, methylmercury eventually redistributes to the skeletal muscles, where it becomes bound to proteins in the muscle tissue (Weiner et al. 2003). In an

extensive review of mercury impacts on fish Weiner and Spry (1996) determined that the binding of assimilated methylmercury to proteins in the skeletal muscles may function as the primary detoxification mechanism for methylmercury in fish. The use of this mechanism reduces exposure of the central nervous system and brain to methylmercury. Because of the eventual redistribution of methylmercury to muscle tissue, the rate of accumulation and exposure time seem to significantly affect the toxicity of methylmercury to fish (Weiner and Spry 1996).

Neurotoxicity seems to be the most probable chronic response of wild fishes to dietary methylmercury, and long-term dietary exposure to methylmercury can cause incoordination, inability to feed, and diminished responsiveness (Weiner and Spry 1996). Other toxicological effects include reproductive impairments (e.g., hatching success, fecundity, and sex steroids), growth inhibition, developmental abnormalities (spinal and jaw deformities), altered behavioral responses (e.g., lethargy, predator response, and aggressiveness), and mortality (as reviewed in Bekvar et al. 1996; Bekvar et al. 2005; Depew et al. 2012; Dillon et al. 2010; Eisler 1987; Weis 2014; Wiener and Spry 1996). Alterations in biochemistry, gene transcription, and tissue histology from exposure to mercury may also be the cause of the deleterious impacts to fish (Moran et al. 2007; Sandheinrich et al. 2011). For example, Moran et al. (2007) found differential gene expression in trout livers collected from two high elevation lakes in Washington. The fish collected from the more polluted lake, primarily higher mercury, exhibited upregulation of genes involved with a number of physiological processes including immune function, stress adaption, reproduction, and metabolism. Surprisingly, even the more contaminated lake fish had low levels of mercury contamination (less than 0.06 µg/g, wet wt., average of 2 years).

Mercury toxicity can have long lasting impacts well after exposure has ended. For example, Fjeld and others (1998) found that sub-lethal methylmercury exposures permanently impaired graylings (*Thymallus thymallus*) three years after the exposure. The 10-day egg exposures that resulted in embryo graylings tissue methylmercury concentrations of 3.8 µg/g (wet wt.) exhibited immediate effects (e.g., delayed hatching, reduce hatching success, and malformed embryos); however, the embryos with body methylmercury concentrations as low as 0.27 µg/g exhibited reduce foraging success (e.g., feeding efficiency and competitive ability) compared to the control group three years after the initial methylmercury exposure. Similarly, Matta and others (2001) observed transgenerational effects with killifish (*Fundulus heteroclitus*) fed methylmercury contaminated food. The maternal transfer of methylmercury to offspring resulted in altered sex ratios and other reproductive abnormalities in the next generation.

Reproductive and early life stage endpoints appear to be some of the most sensitive for fish species, and these adverse effects are typically seen at methylmercury tissue concentrations about 10-fold lower than seen for adult effects (Bekvar et al. 2005; Depew et al. 2012; Dillon et al. 2010; Wiener and Spry 1996). Incubating salmonid eggs will be relatively unaffected by contaminants in the river because vitelline membrane, enveloping layer, and chorion provide defense from metals, pathogens, and xenobiotic chemicals (Finn 2007). Accordingly, the methylmercury accumulated in the eggs will be primarily derived from the maternal fish (Wiener and Spry 1996). Hammerschmidtt and Sandheinrich (2005) concluded that egg methylmercury was primarily derived from the maternal diet during oogenesis because offspring from adults fed mercury before and during oogenesis had similar

concentrations as offspring from adults only fed during oogenesis; however, using stable isotope enriched methylmercury diets, Stefansson et al. (2014) found that both the maternal diet during oogenesis and the female tissue accumulated during preoogenesis contributed mercury proportionally to eggs.

The amount of methylmercury transferred from female to the egg appears to vary depending on contamination level, maternal length, species, etc. For example, the fathead minnow egg concentration percentages increased from 14 to 35% of maternal concentrations with increasing maternal methylmercury diets and maternal concentrations (Hammerschmidtt and Sandheinrich 2005). In another laboratory study with killifish, for the eggs that resulted in methylmercury concentrations above analytical detection limits the percentage of maternal muscle methylmercury concentration in eggs was 0.9% and 5.3%, also increasing with dosage and maternal concentration (Matta et al. 2001). In a field investigation, Johnston et al. (2001) found that egg methylmercury concentrations were 1.1-12% of female muscle concentrations for seven different walleye (*Stizostedion* vitreum) populations. In addition, the percentage of the maternal concentrations varied with maternal length, egg concentrations, maternal liver and muscle concentrations, female length, and population location. Finally, Niimi (1983) investigated the maternal transfer of multiple contaminants in 5 different species collected from Lake Ontario and Erie. The percentage of maternal methylmercury concentrations in eggs averaged: 0.6% for rainbow trout (*0. mykiss*), 1.8% for white sucker (*Catostomus commersoni*), 0.3% for white bass (*Morone chrysops*), 0.4% for smallmouth bass (*Micropterus dolomieui*), and 2.3% for yellow perch (*Perca flavescens*). The field investigations are likely most indicative of typical maternal transfer to eggs the natural environment because these fish reflect the natural bioaccumulation rates, prey methylmercury concentrations, growth rates, etc.

Mercury Objectives

Current numeric water quality objectives or criteria were developed to protect human and other fauna that consume fish and not for the protection of fish themselves. For example, the USEPA promulgated CTR numeric criteria for mercury is for the protection of human health only (40 CFR Part 131). As noted earlier, fish with elevated concentrations of mercury are frequently observed in water bodies that do not exceed the CTR criterion of 0.05 µg/L total mercury. Similarly, water quality objectives developed individually for the San Francisco Bay and the Delta were developed as fish tissue objectives for the protection of human and wildlife consumers of fish (Wood et al. 2010; SFBWQCB 2006). This is in part due to the fact that until recently (within the last decade), the majority of evidence supported that fish were relatively insensitive to mercury toxicity when compared to human and wildlife consumers of fish (Weiner and Spry 1996). For example, Wiener and Spry (1996) concluded that estimated no-observed-effect mercury concentrations for salmonids were 3 µg/g (w.w., whole body), whereas fish tissue mercury concentrations to protect human and wildlife consumers of fish from the San Francisco Bay and Delta is greater than 10-fold lower at approximately 0.2 µg/g (w.w., muscle tissue[2]) (Wood et al. 2010; SFBWQCB 2006).

[2] Muscle tissue (filet) mercury concentrations can be converted to whole-body mercury concentrations using the equation: Log [filet biopsy Hg] = 0.2545 + 1.0623 x Log [whole-fish Hg] (Peterson et al. 2007).

Since 1996, many studies have reported adverse effects to fish species at concentrations lower than the papers reviewed by Wiener and Spry, and there is now evidence that fish species are more sensitive to mercury toxicity than previously thought (Dillon et al. 2010). For example, Beckvar et al. (2005) developed approaches (i.e., simple ranking, empirical percentile, tissue threshold-effect level (t-TEL), and cumulative distribution function) to determine the fish tissue mercury concentrations that would be protective against adverse mercury toxicity using studies that measured mercury tissue concentrations and corresponding biological responses (e.g., reproduction, growth, and behavior). They estimated that a whole-fish mercury concentration of 0.2 µg/g (wet wt.) (filet = 0.33 µg/g wet wt.) would be protective of juvenile and adult fish using the t-TEL method. Using the simple ranking method, Bekvar et al. (2005) estimated that 0.02 µg/g whole-body would be protective of early-life stage fish, which is consistent with the hypothesized higher sensitivity of sublethal effects to embryonic and larval stages mentioned earlier.

Dillon et al. (2010) evaluated mercury effects on fish by developing dose-response curves on lethality-equivalent test endpoints. They found comparable results from dose-response curves as Beckvar et al (2005), and they estimated that a fish mercury concentration of 0.2 µg/g would result in a low (5.5%) injury to juvenile and adult fish. Also consistent with Beckvar et al. (2005), the dose-response curve developed for early-life stage fish predicted an EC50 about seven times lower than the juvenile and adult EC50. Both Beckvar et al. (2005) and Dillon et al. (2010) developed the fish mercury concentrations thresholds using multiple species; however, these thresholds should also be protective of salmonids because the development of the thresholds considers the most sensitive species and endpoints. In addition, there is evidence that salmonid species are less sensitive to the toxicity of dietary methylmercury (Berntssen et al. 2004; Depew et al. 2012).

Mercury Exposures to Salmonids

Due to historical mercury and gold mining and current atmospheric deposition of mercury in California, mercury contamination is a major issue in the Central Valley and Stanislaus River watershed. The potential for fish to bioaccumulate elevated levels of methylmercury clearly exist in the Stanislaus River. For example, fish samples collected from the Stanislaus River since 2000 averaged 0.52 µg/g (wet wt., filet) and ranged between 0.10 and 1.2 µg/g (n = 30, CEDEN 2014b). Many of these fish have mercury concentrations at levels where they could start to exhibit adverse effects. However, these methylmercury concentrations represent Stanislaus River resident fish (e.g., largemouth bass, catfish, and Sacramento sucker) that have bioaccumulated the mercury over many years, whereas anadromous salmonid species typically spend a small proportion of their life cycles in the river. Anadromous salmonid exposure to mercury through bioaccumulation is, for the most part, regulated by food web conditions in the ocean.

Mercury concentration data from returning Chinook salmon and steelhead collected from California's coast, bays, and rivers suggests that salmonid mercury bioaccumulation in the ocean is low (Table 4). For example, the maximum concentration for sampled anadromous Chinook salmon and steelhead, 0.15 and 0.17 µg/g (wet wt., filet), respectively, are below concentrations expected to pose health risks for adult fish. Furthermore, the maternal transfer of mercury to the eggs and larvae likely will not pose health risks to subsequent generations. For example, using the maximum percentage of maternal

transfer from field studies (12%, Johnston et al. 2001), the highest estimated methylmercury concentration in eggs would be 0.02 µg/g (12% x 0.17 µg/g adult). Additionally, the actual amount of transfer for salmonids may be much lower because *O. mykiss* maternal transfer was found to be much lower (0.6%, Niimi 1987).

Table 4
Returning Anadromous Chinook Salmon and Steelhead Tissue Methylmercury Concentrations Collected from California Water Bodies.

Sample Location	Number of Samples	Average Concentration (µg/g, wet wt., filet)	Maximum Concentration (µg/g, wet wt., filet)
Chinook salmon			
American River	5	0.09	0.15
Berkeley	1	0.04	0.04
Coleman Hatchery	5	0.07	0.08
Eel River	1	0.05	0.05
Feather River	5	0.12	0.14
Fort Bragg	4	0.03	0.04
Fort Ross	1	0.04	0.04
Klamath River	1	0.04	0.04
Marin Coast	1	0.07	0.07
Merced River	6	0.09	0.09
Mokelumne River	6	0.11	0.15
Sacramento River	9	0.07	0.09
San Francisco Coastline	1	0.06	0.06
San Pablo Bay	5	0.08	0.11
Chinook salmon Total	51	0.08	0.15
Steelhead			
American River	12	0.07	0.09
Feather River	6	0.09	0.17
Mad River	1	0.10	0.10
Mokelumne River	7	0.10	0.12
Russian River	1	0.09	0.09
Sacramento River	3	0.07	0.10
Steelhead Total	30	0.08	0.17

Notes:
Available fish tissue methylmercury concentration data downloaded from CEDEN on 12/17/2014.

Due to the low mercury bioaccumulation potential in the ocean, anadromous salmonids are likely most at risk to mercury toxicity during freshwater juvenile rearing and out-migrating life stages. However,

since the period of residence in freshwater is relatively short and the food sources have low trophic status (e.g., zooplankton and other invertebrates), juvenile salmonids are not expected to bioaccumulate mercury to levels that would pose survival risks, except in certain environments and rare weather conditions. For example, similar to pesticide exposures salmonids may be exposed to higher contamination of methylmercury in floodplain habitats (Henery et al. 2011; Slotton et al. 2007). Henery et al. (2011) found that juvenile Chinook salmon that reared in Yolo Bypass floodplain accumulated ~3% more methylmercury per day than juveniles that reared in the adjacent Sacramento River. Similarly, Slotton et al. (2007) observed that YOY sculpin collected downstream of salmon restoration zones on the Merced River, Tuolumne River, and Clear Creek had higher (Clear Creek was statistically different) concentrations of tissue methylmercury than sculpins caught upstream of the restorations sites, respectively. However, the average methylmercury concentrations of the fish collected by Henery et al. (2011) and Slotton et al. (2007) were still less than 0.10 µg/g, which are more than 2-fold less than the threshold of concern of 0.2 µg/g.

Further evidence supports low mercury exposure risks to rearing salmonids. For example, juvenile Chinook salmon samples (60-80 mm total length) collected from Marsh Creek in 1995 had average methylmercury concentrations of 0.03 µg/g (wet wt., filet, n= 5, range: 0.01-0.06 (CEDEN 2014c)). Fish rearing in Marsh Creek would likely represent the higher range of mercury bioaccumulation because Marsh Creek has some of the highest average aqueous methylmercury and total mercury concentrations of tributaries to the Delta (Wood et al. 2010).

Unlike the floodplain exposures above, Slotton et al. (2007) did find that the episodic seasonal flooding of dry valley soils in wet years like 2006 could produce environmental conditions that allowed YOY fish to bioaccumulate methylmercury above thresholds of concern. For example, YOY Mississippi silverside (45-75 mm total length) average methylmercury concentrations increased to 0.24 and 0.87 µg/g in the SJR at Vernalis and Cosumnes River, respectively, which represented 4 to 5-fold increases over pre-flooding concentrations. These silversides are of similar size (but may be older) as rearing and out-migrating salmonids, and they may be good representations of the bioaccumulation that could occur in salmonids. In addition, small fish monitoring throughout the region during 2006 (e.g., Yolo Bypass and Suisun Marsh) found similar, but to a lesser extent, elevated concentrations of fish methylmercury. During these types of wet years, migrating salmonids may be exposed to levels of mercury that are risks to their survival throughout their migration to the ocean.

Summary

Overall exposure and risks of mercury toxicity to salmonids is low. Available data suggests that adult anadromous salmonids bioaccumulate methylmercury at levels well below the thresholds of concern (0.2 µg/g, wet wt., whole body). Rearing salmonids may bioaccumulate higher levels of methylmercury in floodplains; however, the risks of survival from mercury are likely offset by the benefits to survival from increased growth rates in these environments (Henery et al. 2011). In addition, the short time (1-12 weeks) that rearing and out-migrating salmonids live in floodplain habitats may preclude them from bioaccumulating high levels of methylmercury, except under extreme conditions (e.g., 2005 and 2006 were both categorized as “wet” WY types in the SJR). However, salmonids that spend an extended time rearing in freshwater (e.g., yearling Chinook salmon or greater than 1 year rearing steelhead) may have

the potential to bioaccumulate methylmercury at levels of concern as migrating smolts. No mercury data were available for Central Valley salmonid yearlings, and yearling mercury bioaccumulation may need further investigation. In addition, further investigation may be necessary to determine if mercury effects on gene regulation are a threat to salmonid health and populations.

Selenium

Selenium is an essential micronutrient for normal animal nutrition; however, selenium can bioaccumulate and biomagnify to levels which are toxic to fish and other wildlife. Selenium can bioconcentrate directly from water through gills, epidermis, or gut; however, like mercury, the primary route of exposure to levels that exhibit toxicological effects is through the food web (Hamilton et al. 2004; Lemly and Smith 1987; Presser and Luoma 2013; USEPA 2014b; Entrix 2009). When dissolved selenium enters the aquatic environment it can: 1) be absorbed or ingested by organisms, 2) bind or complex with particulate matter, or 3) remain in solution (Lemly and Smith 1987). The speciation of dissolved selenium in its three dominant oxidation states (i.e., selenate, selenite, or dissolved organic selenium) is important because the oxidation state of the dissolved form influences the rate of transformations (e.g., oxidation and methylation) that create the particulate form (Lemly and Smith 1987; Presser and Luoma 2013). The uptake of selenate by plants and phytoplankton appears to be slower than the other two (Presser and Luoma 2013).

Ecologically, the first and second mechanisms above are the most important because particulate selenium and selenium associated with plants and phytoplankton are the primary forms that enter the food web (Lemly and Smith 1987; Presser and Luoma 2013; USEPA 2014b). Examples of the mechanisms where selenium is made available for biological uptake include: the oxidation and methylation of inorganic and organic selenium by plant roots and microorganisms, the biological mixing and associated oxidation of sediments that results from the burrowing of benthic invertebrates and feeding activities of fish and wildlife, the physical perturbation and chemical oxidation associated with water circulation and mixing, the oxidation of sediments by plant photosynthesis, and the recycling of particulate phases back into water as detritus or dissolved organic selenium after organisms die and decay (Lemly and Smith 1987; Presser and Luoma 2013). In addition, rooted plants and detrital feeding organisms can input selenium into the food web, even when selenium is absent from the water column (Lemly and Smith 1987).

Selenium has three levels of biological activity in fish: 1) trace concentrations are required for normal growth and development, 2) moderate concentrations can be stored and homeostatic functions maintained, and 3) elevated concentrations can result in toxic effects (Hamilton 2004). Fish exposure to selenium typically follows a biphasic response (e.g., beneficial at low doses; toxic at high doses) (USFWS 2008; Hilton et al. 1980; Lemly and Smith 1987). Toxic effects of selenium to fish typically fall into two categories 1) chronic reproductive (e.g., effects to offspring survival and morphology) and 2) chronic non-reproductive (e.g., adult and juvenile growth and survival) (Lemly and Smith 1987; USEPA 2014b).

Similar to mercury, reproductive function is the most sensitive to selenium toxicity, and the most documented impacts to reproduction are teratogenesis and larval mortality (USEPA 2014b). Often, reproductive failure, whether through effects on adult ovaries or embryonic development, are the first

obvious symptom of selenium contamination, and complete reproductive failure can occur with very little or no tissue pathology or mortality of the adult population (Lemly and Smith 1987). USFWS' (2008) review of selenium impacts to threatened and endangered species in the Delta reported statistically significant increases in pre-swimup mortality and increased percentages of edema and craniofacial deformities in swimup fry with increasing egg selenium concentrations in rainbow trout. In addition, others have reported that fish exposed to selenium exhibit ovaries with necrotic and ruptured egg follicles, anemia and reduce hatch in eggs, or chromosomal aberrations (Eisler 1985). Additional effects of selenium to early life stage fish include deformities that include: lordosis (concave curvature of lumbar and caudal regions of spine), kyphosis (convex curvature of thoracic region of the spine), scoliosis (lateral curvature of the spine); in addition to edema, and brain, heart, and eye problems (Hamilton 2004).

Selenium is transferred from the maternal diet to developing eggs during vitellogenesis, and the embryo is exposed to selenium during yolk absorption (Presser and Luoma 2013; USEPA 2014b). The rate of maternal transfer of selenium to gonadal tissue is much greater than for mercury. For example, Linares-Casenave et al. (2014) found that white sturgeon (*Ancipenser transmontanus*) sampled from the San Francisco Bay and Delta had gonadal tissue selenium concentrations 100 and 200% that of muscle selenium concentrations in previtellogenic and vitellogenic females, respectively. This is compared to the maternal transfer of 0.3-12% of mercury concentrations in gonadal tissues observed in field collected fish (see above). For the development of their draft Aquatic Life Ambient Water Quality Criterion for Selenium, USEPA (2014b) summarized paired maternal and egg-ovary selenium concentrations to estimate conversion factors between tissue concentrations. Individual species conversion factors (maternal muscle>egg-ovary) ranged from 1.0 to 5.8 (i.e., egg concentrations were 100-580% of maternal concentrations), with rainbow trout having the second highest transfer rate (out of 16 species) with a conversion factor of 1.9. The overall high ranking of salmonids continued at the genus level (average *Oncorhynchus = 1.9*) and family level (average Salmonidae = 1.5).

Beyond the reproductive and early life stages, additional effects can occur in fish at later exposures. For example, juvenile rainbow trout fed selenium supplemented diets exhibited reduce growth, higher feed:gain ratio, and higher number of mortalities after 20 weeks of feeding (Hilton 1980). In addition, the juveniles exhibited behavior effects (e.g., feeding avoidance) as well as uncoordinated swimming and sensory deprivation approximately 24-hour priors to mortality. Similarly, Hamilton and Wiedmeyer (1990) found that reduce survival and growth of Chinook salmon were strongly correlated to tissue selenium concentrations in 90-day exposures. As well, selenium exposures to Chinook salmon resulted in reduced survival in the 15-day seawater challenge. Additional effects to fish include: loss of equilibrium, lethargy, contraction of dermal chromatophores, loss of coordination, muscle spasms, protruding eyes, swollen abdomen, liver degeneration, reduction in blood hemoglobin and erythrocyte number, increase in white blood cells, and swollen gill lamellae with extensive cellular vacuolization (Eisler 1985).

In addition to being an essential micronutrient for organisms, selenium has been found to have protective effects against mercury and other metal toxicity (Eilser 1987; USEPA 2014b). However, the mechanism for the antagonistic interactions is not known, the degree of antagonism is highly variable,

and some studies found additive and synergistic interactions with mercury. Laboratory studies by Bjerregaard et al. (2011) suggested that selenium increases the elimination of methylmercury in fish; however, the report acknowledges that other have suggested that selenium may reduce mercury toxicity by redistributing mercury to different tissues or by reducing the assimilation of mercury. Regardless of the mechanism, selenium availability (excess and deficiency) in the aquatic ecosystem must be considered, when considering optimal concentrations in the environment.

Selenium Objectives

USEPA reserved the aquatic life criteria for selenium in the CTR because an USFWS and NMFS biological opinion found that the proposed criteria for selenium may not be protective for threatened and endangered species (USFWS and NMFS 2000). In 2014, USEPA drafted proposed selenium ambient chronic water quality criteria for the protection of aquatic life (Table 5). The proposed criterion allows for multiple matrices to be evaluated (e.g., egg/ovaries, adult fish, and water); and, it takes into consideration that reproduction and early-life stages are the most sensitive to selenium toxicity. In addition, the criterion defaults to tissue selenium concentrations over aqueous selenium concentrations because aqueous concentrations may not reflect the principal exposure routes (e.g., food web and maternal transfer) (Entrix 2009; USEPA 2014b).

The proposed draft criterion for selenium is similar to other criteria and levels of concern determined by others. For example, the Central Valley Water Board water quality objectives for selenium are 5 µg/L and 2 µg/L in the San Joaquin River and Salt Slough, respectively. The draft USEPA aquatic life criterion presents 2 different concentrations because it considers the differences in selenium exposure and bioaccumulation rates of lentic and lotic systems. Based on laboratory toxicity tests, Hamilton and Wiedmeyer (1990) suggested that adverse effects for could occur between 3 and 5 µg/g young salmon (5 g or less) and between 4 and 8 µg/g for older salmon (18 g or more). In a later review by Hamilton (2004), several studies reported aqueous selenium concentrations level of concern in the 1-5 µg/L range, as well as fish whole-body selenium concentration levels of concern in the 2-12 µg/g range. Finally, USFWS (2008) developed statistical models and predicted that 2.5 µg/g would result in a 20% effect in mortality in juvenile Chinook salmon and 2.15 µg/g would result in a 20% reduction in growth in juvenile rainbow trout. In all, salmonid species should be protected against selenium toxicity at tissue concentrations below 2 µg/g (whole body, dry wt.), and tissue concentrations above the 2-4 µg/g range may warrant further investigations to the possible impacts.

Table 5

USEPA Draft National Freshwater Selenium Ambient Water Quality Criterion for Aquatic Life.

Media Type	Fish Tissue		Water Column	
Criterion Element	Egg/Ovary	Fish Whole Body or Muscle	Monthly Average Exposure	Intermittent Exposure

Magnitude	15.2 mg/kg (dry wt.)	8.1 mg/kg whole body or 11.8 mg/kg muscle (skinless, boneless filet) (dry wt.)	1.3 µg/L in lentic aquatic systems 4.8 µg/L in lotic aquatic systems	$WQC_{int} = \frac{WQC_{30\text{-}day} - C_{bkgrnd}(1 - f_{int})}{f_{int}}$
Duration	Instantaneous measurement	Instantaneous measurement	30 days	Number of days/month with an elevated concentration
Frequency	Never to be exceeded	Never to be exceeded	Not more than once in three years on average	Not more than once in three years on average

Notes:
From External Peer Review Draft Aquatic Life Ambient Water Quality Criterion for Selenium - Freshwater 2014 (USEPA 2014b). These draft criteria are presented to give a relative magnitude of selenium levels above which could pose risks to aquatic life. In addition, the criteria are presented as an example of the type of approach that could be used to assess selenium impacts to aquatic life. The criteria have yet to be peer review, and they have not been promulgated by USEPA.

Selenium Exposures to Salmonids

As describe previously, the two life stages that anadromous salmonids would be the most at risk to selenium exposure are 1) reproductive and early-life stages (e.g., ovary, egg, and larvae) and 2) rearing and out migrating stages. Possible exposure of these life stages in the Stanislaus River as well as in their downstream migration will be described in this section.

Reproductive and early-life stages of anadromous salmonids should not be adversely impacted by selenium contamination in the Stanislaus River. First, based on tissue selenium concentrations for returning adult anadromous Chinook salmon and steelhead collected from California, low levels of selenium would be transferred from females to eggs. For example, the maximum tissue selenium concentration in Chinook salmon and steelhead samples was 1.2 µg/g (whole body, dry wt., Table 6), which would, consequently, result in an estimated maximum concentration of 3 µg/g in eggs (assuming a conversion factor of 1.9 for *Oncorhynchus*). This is well below the draft aquatic life criteria proposed by USEPA (2014b) (Table 5). Second, incubating eggs should be relatively unaffected by selenium contamination in the river, in part due to their resistance to contaminants and the low levels of selenium concentrations in the river (Finn 2007). For example, the maximum aqueous total selenium concentration observed in the Stanislaus River between 1998 and 2005 was 0.45 µg/L (n = 65, CEDEN 2014a), and adverse effects to incubating eggs haven't been observed until aqueous selenium concentrations exceed 25 µg/L (Hamilton and Wiedmeyer 1990; Hodson et al. 1980; Lemly and Smith 1987).

Table 6

Returning Anadromous Chinook Salmon and Steelhead Tissue Selenium Concentrations Collected from California Water Bodies.

Sample Location	Number of Samples	Average Concentration (µg/g, dry wt. whole body)	Maximum Concentration (µg/g, dry wt. whole body)
Chinook salmon			
Eel River	1	0.45	0.45
Fort Bragg	4	0.71	0.77
Fort Ross	1	0.66	0.66
Klamath River	1	0.79	0.79
Marin Coast	1	0.74	0.74
Mokelumne River	1	0.42	0.42
Sacramento River	1	0.92	0.92
San Francisco Coastline	1	0.66	0.66
Chinook salmon Total	11	0.68	0.92
Steelhead			
Mad River	1	0.93	0.93
Mokelumne River	1	0.90	0.90
Russian River	1	1.15	1.15
Steelhead Total	3	0.99	1.15

Notes:
Available fish tissue selenium concentration data downloaded from CEDEN on 12/17/2014.
Calculated from wet wt. assuming 72% moisture (Hamilton and Wiedmeyer 1990)

The low level of selenium contamination in the Stanislaus River water continues through the food web. For example, Stanislaus River largemouth bass and channel catfish tissue selenium concentrations are low (Table 7). There is few data; however, the composite analyses were from species that are from the pelagic food web as well as from the benthic food web. The use of both types of species (e.g., pelagic and benthic) is the preferred method to characterize overall selenium contamination in a waterbody (Davis et al. 2013). Juvenile salmonids should not bioaccumulate higher levels than resident largemouth bass or channel catfish because of their short residence time and lower trophic level diet. In addition, salmonids that rear for longer periods (e.g., spring-run or steelhead yearlings) in the Stanislaus River should be at little risk of selenium toxicity because they too should have lower bioaccumulation rates than top trophic level black bass or long lived benthic species.

Table 7

Composite Fish Tissue Selenium Concentrations Collected from the Stanislaus River

Fish Species	Number of Fish in Composite	Tissue	Se Concentration (µg/g, dry wt.)
Largemouth bass	5	muscle	1.4
Channel catfish	10	liver	3.6
Channel catfish	4	liver	2.9

Notes:
Available fish tissue selenium concentration data downloaded from CEDEN on 10/09/14.
Calculated from wet wt. assuming 72% moisture (Hamilton and Wiedmeyer 1990)

Rearing and migrating salmonids would have greater risks of selenium toxicity in the San Joaquin River and San Francisco Bay because they have known contaminations of selenium. For example, agricultural discharges in the upper San Joaquin River watershed from the Grasslands area resulted in elevated selenium levels in the river, and the river frequently exceeded water quality criteria for total selenium (5 µg/L, CVRWCB 2001). In addition to the agricultural discharges from the upper watershed, the San Francisco Bay has received selenium contamination from effluent discharges from oil refineries, as well as from the Sacramento River (Presser and Luoma 2006). Furthermore, white sturgeon as well as other predatory fish and birds in the Bay-Delta have been identified as bioaccumulating high levels of selenium that pose immediate risks to their health (Linares-Casenave et al. 2014; Presser and Luoma 2006).

Fortunately, regulatory programs have reduced selenium discharges into surface waters (McCarthy and Grober 2001; Presser and Luoma 2006; SFBRWQCB 2013). In 1996, the Central Valley Water Board developed a control program to control selenium from agricultural discharges (McCarthy and Grober 2001). Since the adoption of the San Joaquin River Selenium TMDL, the maximum aqueous total selenium concentration observed in the San Joaquin River at Vernalis was 3.7 µg/L (n = 510, CEDEN 2015). Furthermore, none of the 30 sites monitored in the Delta or the San Francisco Bay since 2001 exceeded dissolved selenium concentration greater than 1.01 µg/L (n = 231, CEDEN 2015). The concentrations of selenium in the water is low compared to the water quality objectives that have been adopted by the Central Valley Water Board, as well as the draft criteria being proposed by USEPA (McCarthy and Grober 2001; USEPA 2014b).

However, aqueous selenium concentration is only one factor regulating selenium bioaccumulation and exposure, and the type food web has been found to be the major factor determining the bioaccumulation rates and exposure of aquatic life to selenium toxicity (Lemly and Smith 1987; Presser and Luoma 2013; Stewart et al. 2004; USEPA 2014b). Different predators in the same water body can have highly variable selenium concentrations depending on whether they consume from a clam based food web (benthic) or an insect based food web (pelagic) (Presser and Luoma 2013). Bivalves depurate selenium slower than similar trophic level insects and crustaceans, so they can have 2-5-fold higher concentrations of selenium (Stewart et al. 2004). These elevated concentrations of selenium continue through the food web, and benthic predators like sturgeon and splittail bioaccumulate high levels of selenium (Stewart et al. 2004). Fortunately, rearing and out migrating salmonids consume invertebrates from the pelagic food web, so they are less susceptible to selenium exposure and toxicity (Presser and Luoma 2013).

Summary

Selenium is a natural essential nutrient; however, anthropogenic activities have increased its concentration in some water bodies in the Central Valley and Bay-Delta to levels which cause toxic

effects to aquatic organisms and the terrestrial organisms that rely these organisms. The primary route of exposure to selenium is through the diet and food web, and the food web that poses the highest risk of selenium toxicity is the benthic or clam based food web. Fortunately, salmonids consume organism from the pelagic or insects food web while rearing and out migrating to the ocean. In addition, the low selenium concentrations in returning adult anadromous salmonids suggest that selenium bioaccumulation in the ocean is also low. However, salmonids are some of the most sensitive fish species, so toxic effects may occur at levels which do no cause adverse effects in other species (Presser and Luoma 2013). If salmonid tissue selenium concentrations approach the level of concern range of 2-4 µg/g, then further investigation may be necessary to determine if selenium toxicity is causing adverse impacts to individuals or populations of Chinook salmon or steelhead.

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